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Electronic paper

Flexible active-matrix electronic ink display

Ultrathin, flexible electronic displays that look like print on paper are of great interest[1–4] for application in wearable computer screens, electronic newspapers and smart identity cards. Here we realize the fabrication of such a display on a bendable active-matrix-array sheet. The display is less than 0.3 mm thick, has high pixel density (160 pixels × 240 pixels) and resolution (96 pixels per inch), and can be bent to a radius of curvature of 1.5 cm without any degradation in contrast. This use of electronic ink technology on such an ultrathin, flexible substrate should greatly extend the range of display applications.

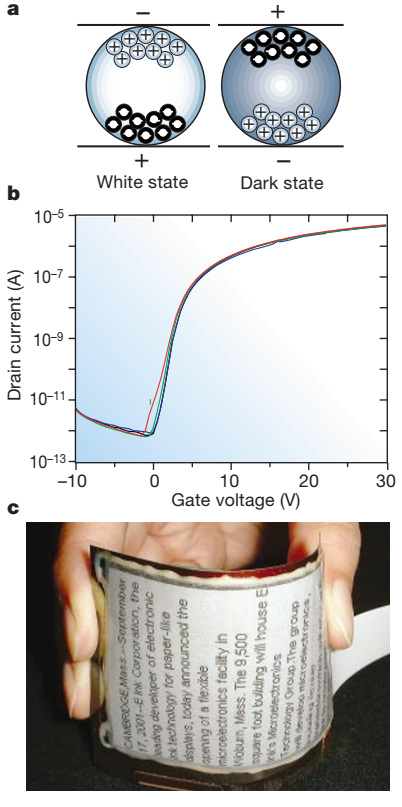

Figure 1 Flexible active-matrix electronic-ink displays. **a,** Operating principle of electronic ink. The relative movement of negatively charged black and positively charged white particles inside their microcapsules is controlled by the direction of the applied voltage. **b,** A backplane thin-film transistor measured *in situ* under compressive stress. The transistor is bent to three different radii of curvature: green, 2.0 cm (0.19% strain); blue, 1.3 cm (0.29% strain); and red, 1.0 cm (0.38% strain). The thin-film transistor has identical characteristics when measured without bending (black curve) and at a radius of curvature of 2.0 cm; degradation is minimal even at 1.0 cm. Results were similar under tensile stress. **c,** Text image shown on a bent display whose resolution is 96 d.p.i. and which has a white-state reflectance of 43% and a contrast ratio of 8.5:1.

Thin (0.4-mm) but inflexible liquid-crystal displays have been made on plastic by using a diode-matrix array[5] and an amorphous-silicon, thin-film transistor (TFT), active-matrix array[6]. To create a flexible display, we used a TFT array (backplane) with microencapsulated electrophoretic material (electronic ink)[7], which consists of millions of microcapsules containing charged pigment particles in a clear fluid. A negative voltage applied to the top surface causes the positive white particles to move to the top of the capsule and the surface to appear white; reversing the electric field causes the negative black particles to appear at the top surface and create a dark spot (Fig. 1a).

We used a 75-μm-thick steel-foil substrate to build the TFT backplane because steel foil is lightweight, mechanically stable and compatible with existing fabrication processes for active-matrix liquid-crystal displays[8,9]. Before the array fabrication, an insulating layer was applied onto the foil to render the substrate passive. The amorphous-silicon TFTs were made in the bottom-gate, back-channel etch configuration. The gate and source/drain metal were deposited by sputtering. A ductile composite of aluminium and refractory metal was used for the gate metal to enhance the backplane's flexibility.

Silicon nitride, amorphous silicon and a doped amorphous-silicon layer were deposited as the gate insulator, the channel and the contact layer, respectively, by plasma-enhanced chemical-vapour deposition. The metal, semiconductor and insulator layers were patterned by photolithography. The display was made by laminating a sheet of electronic ink onto the backplane. The electronic ink consists of a layer of electrophoretic microcapsules and a polymer binder, coated onto a polyester/indium–tin oxide (common electrode) sheet. The total display thickness is less than 0.3 mm.

A typical TFT has a threshold voltage of 4.0 volts and a linear mobility of 0.50 $cm^2 V^{-1} s^{-1}$. The drain off current is about 1.0 pA at 10 V drain voltage. The current on/off ratio is 5×10^6, which is sufficient for high-resolution displays. The TFT performance does not degrade after first being bent for 120 seconds around a cylinder that is 2 mm in radius (1.9% strain) and then released.

We also measured TFTs *in situ* under compressive stress at three radii of curvature (Fig. 1b). Because steel has a large Young's modulus, our selection of a thin substrate decreases the distance of the TFT circuit from the display neutral plane[10], reducing the in-plain strain of the circuit. As a result, the display can be repeatedly bent 20 times to a radius of curvature of 1.5 cm without any degradation.

Bias temperature stress on the TFT backplane was performed at a gate voltage of 25 V and up to 80 °C. The results indicate that the flexible backplane has a threshold voltage shift comparable to that of conventional glass TFT backplanes in laptop computers, and possibly a similar reliability and lifetime. The row electrode is driven between 0 and 24 V, and the column electrode is driven between 0 and 20 V.

Figure 1c shows the bent display of a text image of 96 d.p.i. resolution; the display has a viewing angle of almost 180°. The ink-switching speed is 250 ms, which is sufficient for electronic paper. For wearable computers, a reduction to 15 ms would be required for video-rate switching; in addition, the substrate thickness would need to be reduced for foldable displays. We suggest that electronic ink combined with flexible amorphous-silicon active-matrix backplanes will provide a viable pathway to 'e-paper' and wearable computer screens.

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Comparative genomics

Insecticide resistance in mosquito vectors

Resistance to insecticides among mosquitoes that act as vectors for malaria (*Anopheles gambiae*) and West Nile virus (*Culex pipiens*) emerged more than 25 years ago in Africa, America and Europe; this resistance is frequently due to a loss of sensitivity of the insect's acetylcholinesterase enzyme to organophosphates and

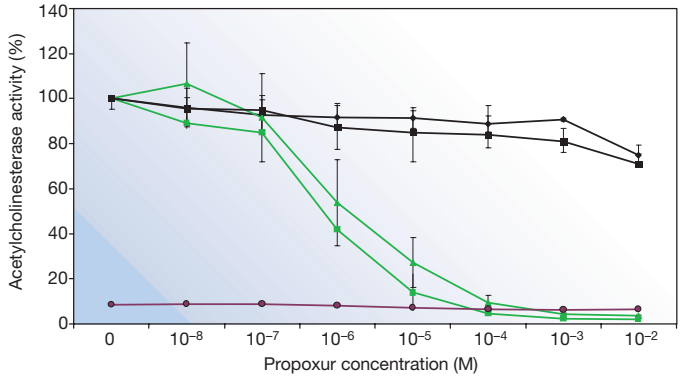

Figure 1 Residual acetylcholinesterase activity of susceptible (green squares) and resistant (black diamonds) mosquitoes assayed in homogenates and lysates from transfected S2 cells in the presence of increasing concentrations of the carbamate insecticide Propoxur. S2 cells were transfected with the recombinant pAc5.1/V5-His vector (Invitrogen) either alone (purple circles) or with expression of either sensitive acetylcholinesterase-1 (green triangles) or insensitive G119S-mutant enzyme (black squares). Residual enzyme activity was assayed after incubation with Propoxur for 15 min (ref. 5). Three independent experiments were carried out using different volumes of cell lysate.

carbamates[1]. Here we show that this insensitivity results from a single amino-acid substitution in the enzyme, which we found in ten highly resistant strains of *C. pipiens* from tropical (Africa and Caribbean) and temperate (Europe) areas, as well as in one resistant African strain of *A. gambiae*. Our identification of this mutation may pave the way for designing new insecticides.

Acetylcholinesterase terminates synaptic transmission by hydrolysing the neurotransmitter acetylcholine; its inactivation by insecticides leads to paralysis and death. Mosquitoes, however, show widespread and strong resistance to this type of insecticide. They have two genes that encode different isoforms of acetylcholinesterase: *ace-1*, which has no homologue in the fruitfly *Drosophila melanogaster* and is closely linked to resistance in *C. pipiens*; and *ace-2*, a homologue of the unique *Drosophila ace* gene[2]. The generally mild insensitivity of acetylcholinesterase-2 in *D. melanogaster* is due to the combined weak effect of several mutations[3].

To identify mutations involved in resistance in mosquitoes, we determined the complete *ace-1* messenger RNA coding sequence of two *Culex pipiens* strains: one susceptible and one resistant (results not shown). *C. pipiens ace-1* encodes a putative 702-amino-acid protein, which is 81% identical to its *A. gambiae* homologue and 39% identical to *D. melanogaster* acetylcholinesterase-2. Complementary DNAs from the susceptible and resistant strains differ at 27 nucleotide positions, only one of which generates an amino-acid substitution: the GGC (glycine) codon at position 119, according to the nomenclature for *Torpedo* acetylcholinesterase[1], is replaced by an AGC (serine) codon in resistant mosquitoes (mutation G119S).

From three-dimensional modelling, we find that this mutated residue lies within the active 'gorge' of the enzyme, close to the catalytic site and abutting the oxyanion hole (results not shown). To evaluate the biochemical effect of the mutation *in vitro*, we assayed the catalytic properties and insecticidal sensitivity of wild-type and mutant recombinant acetylcholinesterase-1 that was expressed in S2 *Drosophila* cells. The G119S mutant showed the same insensitivity to Propoxur insecticide as resistant-strain acetylcholinesterase-1 (Fig. 1). A single mutation in *ace-1* must therefore be responsible for the insensitivity of the enzyme.

To determine whether the G119S mutation is present in other *C. pipiens* strains with insensitive acetylcholinesterase, we sequenced exon 3 of *ace-1* in several resistant and susceptible strains derived either from the temperate or the tropical/subtropical form of the *C. pipiens* species complex (*C. p. pipiens* and *C. p. quinquefasciatus*, respectively). All of the resistant strains carried the G119S substitution, regardless of their origin. Moreover, although 23 nucleotides were polymorphic, a unique haplotype was found to be associated with the resistance within each subspecies (see supplementary information). This indicates that the same G119S mutation has occurred independently at least twice in *C. pipiens*, once in each subspecies.

We also investigated the recent emergence of insensitive acetylcholinesterase in the main African malaria vector *Anopheles gambiae*[4], with the use of the *ace-1* genomic sequences of a resistant (YAO) and a susceptible (KISUMU) strain. The coding sequences differed at 18 nucleotide positions, two of them being non-synonymous. In the YAO strain, one mutation that resulted in the replacement of a valine residue by alanine in the amino-terminal region has no equivalent in *Torpedo* acetylcholinesterase and did not seem to affect the enzyme's catalytic properties (results not shown). The other was the same G119S substitution as in *C. pipiens* (results not shown), indicating that this single point mutation has occurred independently at least three times in the *ace-1* gene: twice in the *C. pipiens* complex and once in *A. gambiae*.

The discovery of the *ace-1* mutation that is responsible for insecticide resistance in mosquitoes opens the way to new strategies for pest management. The development of new insecticides that can specifically inhibit the G119S mutant form of acetylcholinesterase-1 will be crucial in overcoming the spread of resistance.

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COMMUNICATIONS ARISING

Nitrogen storage

UV-B radiation and soil microbial communities

Soil microorganisms regulate the supply of nitrogen to plants and so are important controllers of plant productivity and ecosystem carbon sequestration. Johnson *et al.*[1] report that exposure of a subarctic heath ecosystem to increased ultraviolet-B (UV-B) irradiation causes a drastic decline in the mass ratio of C:N in soil microorganisms, which would increase the amount of nitrogen stored in the microbial biomass and possibly alter the availability of nitrogen to plants. However, we argue that some of the authors' microbial C:N data are unrealistic, possibly because of an artefact of the technique used to measure microbial carbon and nitrogen concentrations. As a result, there is little reason to suppose that increased exposure of ecosystems to UV-B radiation will influence microbial nitrogen storage, plant nitrogen availability or rates of carbon sequestration.

Johnson *et al.* calculated an average microbial C:N ratio of 36.2 in control plots,

Archaeology

Sharp shift in diet at onset of Neolithic

The introduction of domesticated plants and animals into Britain during the Neolithic cultural period between 5,200 and 4,500 years ago is viewed either as a rapid event[1] or as a gradual process that lasted for more than a millennium[2]. Here we measure stable carbon isotopes present in bone to investigate the dietary habits of Britons over the Neolithic period and the preceding 3,800 years (the Mesolithic period). We find that there was a rapid and complete change from a marine- to a terrestrial-based diet among both coastal and inland dwellers at the onset of the Neolithic period, which coincided with the first appearance of domesticates. As well as arguing against a slow, gradual adoption of agriculture and animal husbandry by Mesolithic societies, our results indicate that the attraction of the new farming lifestyle must have been strong enough to persuade even coastal dwellers to abandon their successful fishing practices.

Stable carbon isotopes in human bone collagen act as indicators of past dietary intake[3] because marine and terrestrial dietary proteins leave different 'signatures'[4]. Consumption of cereal crops that use the C_3 photosynthetic pathway and of farmed animals should result in a 'terrestrial' bone-collagen carbon-isotope signature ($\delta^{13}C = -20 \pm 1‰$, where $\delta^{13}C$ represents the $^{13}C/^{12}C$ ratio), whereas marine foods give a much higher ^{13}C content ($\delta^{13}C = -12 \pm 1‰$).

Archaeological evidence for the use of marine foods during the British Mesolithic is limited because very few coastal sites survived the rising sea levels of the more recent Holocene epoch. Some of the best-known exceptions are the late Mesolithic shell middens of western Scotland[5,6]. Other areas of Atlantic Europe, most notably southern Scandinavia and Brittany, present strong archaeological and isotopic evidence of marine-based economies at this time.

We have measured and collated[6–8] the carbon-isotope values of bone collagen of 164 early Neolithic (5,200–4,500 yr BP) and 19 Mesolithic (9,000–5,200 yr BP) British humans. The Neolithic sample is derived from a range of contexts, including causewayed enclosures, chambered tombs, caves and stray finds, from both inland and coastal locations. Although individuals from inland and putative 'élite' contexts are more prominently represented, the results from all of these contexts are unanimous.

Figure 1 shows that, with few exceptions, individuals living near the coast in the Mesolithic show a moderate-to-strong marine isotope signal (for four humans from two inland sites, $\delta^{13}C = -19.6 \pm 0.8$; for fifteen humans from eight coastal sites, $\delta^{13}C = -16.2 \pm 2.8$), and that all of the Neolithic humans show a strongly terrestrial isotope signal (for 99 humans from 25 inland sites, $\delta^{13}C = -20.7 \pm 0.7$; for 68 humans from 19 coastal sites, $\delta^{13}C = -20.8 \pm 0.7$). These data are comparable with results obtained in Denmark, which also show a rapid dietary change in humans between the Mesolithic and Neolithic at about the same time[9,10].

From our findings, we conclude that there was a sudden and marked dietary shift associated with the onset of the Neolithic period in Britain, arguing against a gradual uptake of domesticated plants and animals into Mesolithic society[2]. Marine foods, for whatever reason, seem to have been comprehensively abandoned from the beginning of the Neolithic in Britain.

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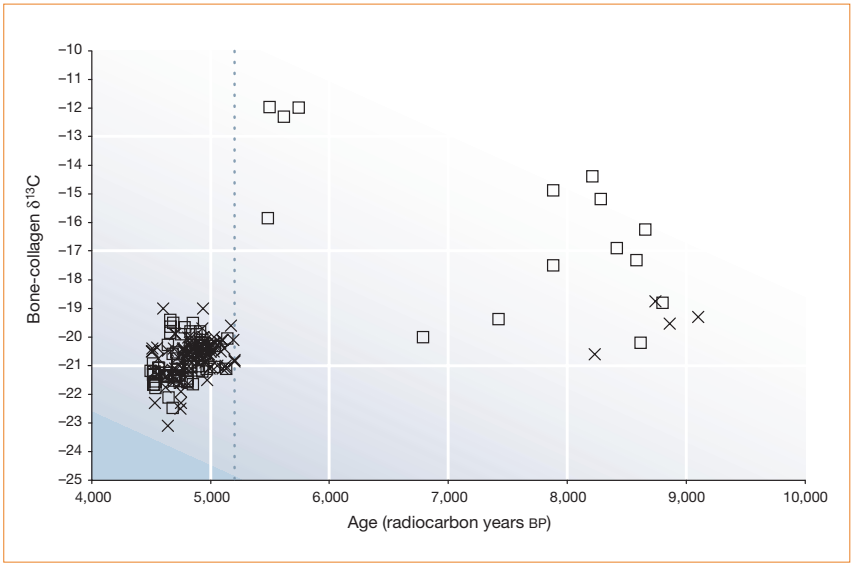

Figure 1 Bone-collagen carbon-isotope ratios and radiocarbon ages of 183 Mesolithic and Neolithic humans from coastal (that is, living within 10 km of contemporary coastline; squares) and inland sites (crosses) in Britain. There is a sharp change in the carbon-isotope ratio at around 5,200 yr BP (about 4,000 calendar yr BC; dotted line) from a diet consisting of marine foods to one dominated by terrestrial protein. This period coincides with the onset of the Neolithic period in Britain. Because of uncertainties in the size of the marine reservoir effect on radiocarbon dates, we have not attempted to calibrate the data here; however, for individuals with typical marine $\delta^{13}C$ values (about −12‰), radiocarbon dates should be corrected by roughly 400 radiocarbon years.

corrigendum

Insecticide resistance in mosquito vectors

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Nature **423,** 136–137 (2003)

It has been drawn to our attention (I. Gould and P. A. Zimmerman) that the numbering of the amino-acid sequences and exon presented in our communication differs from that of the corresponding EMBL/GenBank entries. Because mosquito proteins differ in length, the first published acetylcholinesterase three-dimensional structure (from *Torpedo californica*) was used to number structurally identical residues. The glycine residue whose substitution by serine confers insecticide insensitivity was therefore numbered 119, whereas it corresponds to amino acids 247 and 280 of *Culex pipiens* (entry CAD33707) and *Anopheles gambiae* (entry CAD56157) proteins, respectively. In *Anopheles*, this position lies within the third coding exon (exon 5). Our conclusions are not affected by this inconsistency.

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Vision

Steady-state misbinding of colour and motion

When you see a red ball rolling across the floor, the ball's redness, roundness and motion appear to be unified and inseparably bound together as features of the ball. But neurophysiological evidence indicates that visual features such as colour, shape and motion are processed in separate regions of the brain[1]. Here we describe an illusion that exploits this separation, causing colour and motion to be recombined incorrectly while a stable stimulus is being viewed continuously.

The illusion is seen in stimuli containing two sheets of random dots, where one sheet is moving up and one is moving down. The sheets contain dots of two colours such that the central and peripheral portions of the stimuli combine colour and motion in opposite fashions (Fig. 1a). On the upward-moving sheet, dots in the centre are red and dots in the periphery are green. On the downward-moving sheet, dots in the centre are green and dots in the periphery are red. (For movie, see supplementary information.)

Observers gazing at the centre of the display perceive peripheral dots erroneously: they 'bind' colour and motion in the wrong combination. The entire display therefore appears to be covered by a sheet of red dots that are moving upwards and a sheet of green dots that are moving downwards.

To quantify this effect, observers were asked to report whether the majority of peripheral red dots were moving up or down over a series of trials in which we varied the percentage of peripheral dots moving in either direction. In control trials, where the central portion of the stimulus was omitted, responses of observers ($n = 5$; 3 naive) roughly followed the physical stimulus (Fig. 1b, black line). But when the central portion was present, responses shifted markedly, and in the same direction as the central dots (Fig. 1b, red and green lines; MANOVA, $F(10,16) = 35.2$, $P < 0.001$).

To ensure that observers' responses were reflecting their perception and not a simple bias to respond to the central dots, we generated stimuli in which one peripheral region matched the centre and the other contained the opposite motion. Observers ($n = 4$; 3 naive; all from the first experiment) perceived both sides to move with the centre, performing poorly in reporting which side moved the other way (mean ± s.e.m.: 47 ± 8% correct; sensitivity measure $d' = -0.12 \pm 0.38$). On the other hand, they performed well in reporting which side was which when the stimulus centre was blank (86 ± 2.5% correct; $d' = 1.5 \pm 0.2$, paired t-test $P < 0.5$).

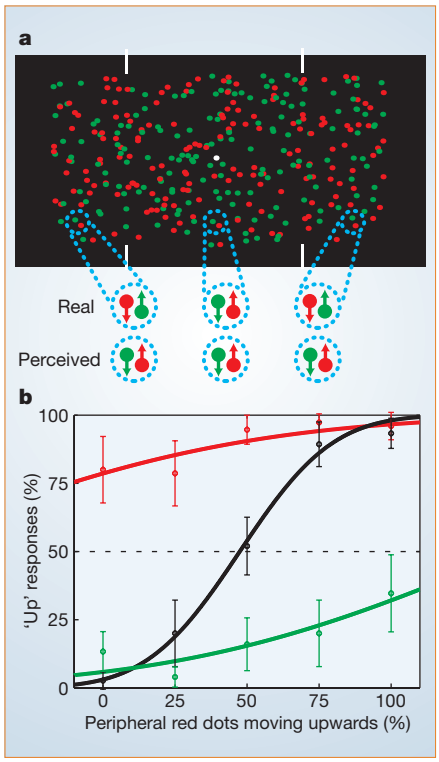

Figure 1 Steady-state misbinding stimulus and observers' reports. **a,** Sample transparent motion stimulus. Two sets of random dots move rigidly in opposite directions. 'Real' circles show details of the physical stimulus. In the upward-moving surface, dots near fixation are red, and dots beyond 6° of horizontal eccentricity are green; in the downward-moving surface, the reverse coloration applies. White bars marked the transition line. 'Perceived' circles show details of the illusory percept. Observers incorrectly pair colour and motion in the periphery. Across the entire stimulus, upward-moving dots appear red and downward-moving dots appear green, forming two homogeneous surfaces of dots moving in opposite directions. **b,** Proportion of trials in which observers judge that most red dots beyond the white bars are moving upwards, plotted as a function of the actual percentage of red dots moving upwards; time to view stimuli was unlimited. In control trials (black line), the central region is blank, and responses follow the stimulus. When the central region contains upward-moving red dots and downward-moving green dots (red line), responses shift predominantly to 'up'. When the central region contains the opposite motion (green line), responses shift predominantly to 'down'.

We propose that the illusion described here is the result of an ambiguity-resolving mechanism. Because vision is clearest at the point of gaze, it would normally be advantageous to use information from the centre to resolve peripheral ambiguity if the physical source is likely to be a common uniform surface. Uniform surfaces are strongly implied by the contiguous and precisely equal values of colour and motion present across this stimulus. Peripheral features are bound in such a way as to accord with the usually more reliable centre, although in this case the binding is mistaken.

Previous investigations of basic visual feature binding required the use of stimuli with brief presentation times or rapidly changing features to induce errors or inefficiencies[2–4]. The illusion presented here occurs despite fully attentive and continuous viewing, avoiding the common confounds of memory, expectation and task strategy[5]. It solidifies the evidence for the existence of a binding problem and provides a substrate for neurophysiological investigation.

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Corrigendum

Insecticide resistance in mosquito vectors
M. Weill, G. Lutfalla, K. Mogensen, F. Chandre, A. Berthomieu, C. Berticat, N. Pasteur, A. Philips, P. Fort, M. Raymond
Nature **423,** 136–137 (2003).
It has been drawn to *Nature*'s attention that M. W., N. P., P. F. and M. R. are named as inventors on two patent applications relevant to this work (FR20020007622 20020620, filed in June 2002; and FR200200213799 20021105, filed in November 2002), which should therefore have been declared as competing financial interests.

brief communications arising online
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Evolutionary biology: Lamprey *Hox* genes and the evolution of jaws
Y. Takio, M. Pasqualetti, S. Kuraku, S. Hirano, F. M. Rijli, S. Kuratani (doi:10.1038/nature02616)